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THE FACTORS INFLUENCING THE PROMINENCE OF THE DICROTIC WAVE.

By THOMAS LEWIS *of University College, London.* (Fifteen Figures in the Text.)

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THE FACTORS INFLUENCING THE PROMINENCE OF THE DICROTIC WAVE.

By THOMAS LEWIS
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THIS contribution is based on the results of a prolonged series of observations on the pulse, physiological and pathological. The instruments employed have been Dudgeon's Sphygmograph, Mackenzie's Polygraph and C. J. Martin's modification of the Riva-Rocci Sphygmomanometer. The observations have been on man only. I take this opportunity of expressing my thanks to my colleagues in residence at University College Hospital, and to those members of the resident staff who since my term of office ended, have given me every facility in the prosecution of such enquiries, as I have considered of interest and service. My thanks are also due to Mr H. L. Lewis, R.E., for the valuable assistance he has given me in these investigations.

For the purposes of this communication it is unnecessary to enter upon a discussion of the complex subject of the origin of dicrotic wave; the observations will be confined as far as possible to the factors productive of an increased wave. The arguments involved and the conclusions derived from them are compatible with the now generally accepted hypothesis, that the dicrotic wave is of central origin, and takes place as the result of a reflection of a pressure wave from the aortic valves and neighbouring walls of the larger vessels. Before entering upon a discussion it will be advisable to record the observations made upon dicrotism during the course of these investigations.

In an earlier communication, the work of Roy and Adami was referred to, and the conclusions which they drew from Valsalva's experiment were defended. In the communication referred to it was proved that a pulse may become more dicrotic in spite of a considerable rise of blood-pressure. Moreover the dicrotism produced during a period of the experiment when intrathoracic pressure is still high, is as a general rule of greater degree than that which occurs on the release of pressure. A similar increase of dicrotism is often observable at the

termination of a deep inspiration[1], when the output of the heart is presumably increased, and the mean pressure raised (Fig. 1).

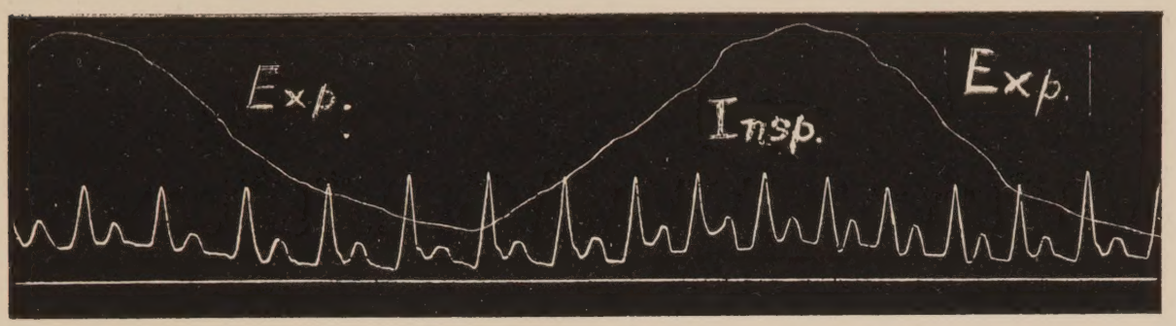


Fig. 1.

There are a good many clinical observations which have a close bearing on the subject of the relation of pressure to dicrotism. Galabin described a hard pulse in sthenic fever, markedly dicrotic and bearing high pad pressure. Roy and Adami noted increased dicrotism in an individual in whom there was no reason to suspect lowered pressure. Equally significant cases are to be found in the writings of Mahomed. The methods adopted by these observers, in arriving at an estimate of blood-pressure, are now recognised as inaccurate. Consequently it has been thought desirable to study the relation more carefully, and with the accurate modern instruments. It has been found that pulses dicrotic throughout may occur with systolic pressures varying from 80—125 mm. Hg., and that dicrotic pulses of this character are met with commonly at all intermediate pressures. The pulses here referred to are those which give a graphic record, exhibiting primary and dicrotic waves only. Examples of such pulses are given in Figs. 2, 3 and 4. Fig. 2 is from a boy aged 11, suffering from mitral stenosis, with poor compensation, pulse rate 240 per minute, systolic blood-pressure 80 mm. Hg. Fig. 3 is from a boy aged 16, suffering from double mitral disease, with fair compensation, systolic blood-pressure 90 mm. Hg. Fig. 4 is from a lad aged 17, admitted for acute pleurisy, systolic blood-pressure 120 mm. Hg. Fig. 5 is of special interest; these two tracings were taken from the same patient as that shown in Fig. 4, the left-hand tracing 10 days after the aspiration of the fluid from the chest, when the systolic blood-pressure was 97 mm. Hg. In this tracing

[1] The observations dealing with influence of inspiration and expiration on the pulse have given very variable results. Inspiration has sometimes given a mean rise, and sometimes a fall. In the tracing given the systolic line and diastolic line show converse curves, a very common phenomenon. The subject requires reinvestigation.

dicrotism appears decreased in spite of a considerable fall of pressure, and a predicrotic wave has appeared. The right-hand tracing, taken 18 days later, during recovery, corresponded to a pressure of 108 mm. Hg. The tracing is practically a normal one. Fig. 6 is from a man admitted to hospital with acute Bright's disease complicating interstitial nephritis, blood-pressure 160 mm. Hg. He was bled for threatening uræmia and the first two tracings were taken directly before and after a venesection of 20 oz. The first tracing, blood-pressure 160 mm., shows two dicrotic beats, one displaced downwards and presumably at a slightly

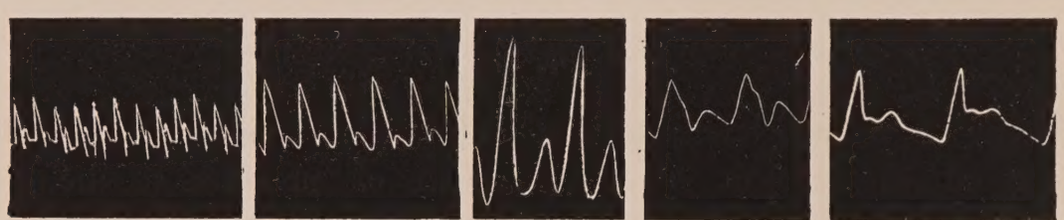


Fig. 2. Fig. 3. Fig. 4. Fig. 5.

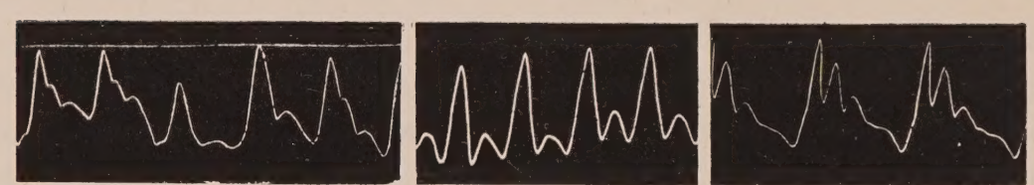


Fig. 6.

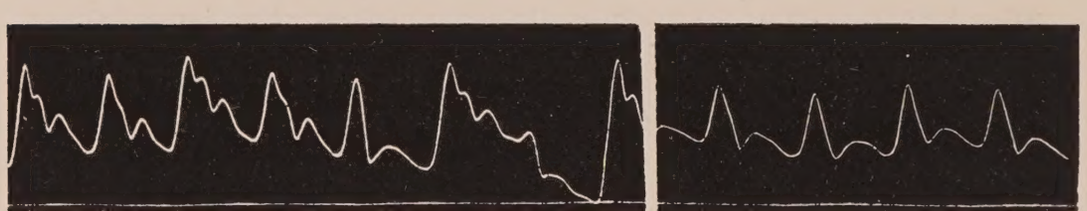


Fig. 7.

lower mean pressure. During the venesection numerous tracings were taken, but the first to show pure[1] dicrotism was not obtained until the pressure fell to 125 mm. The tracing is the second in Fig. 6. The third tracing in this figure shows the pulse three days later, when all the waves were well marked, the blood-pressure being 150 mm. A double wave was palpable, though it was not possible to identify the second with the predicrotic or dicrotic. Fig. 7 shows tracings from a somewhat similar case. The man, suffering from chronic interstitial nephritis, was bled to the extent of 20 oz. for uræmic fits ; before the

[1] By a purely dicrotic pulse is signified one which shows two waves only.

venesection the pulse exhibited the characters shown in the first tracing (blood-pressure 170 mm.). The pulse first became purely dicrotic when the pressure reached 110 mm.

The two series of tracings are of special interest, for first they contain all the transitions from the types of curve usually recognised as representing high and low blood-pressures[1]. Such transitions from high to low pressure curves are figured by Marey, Kries, Tigerstedt, Galabin, Roy and Adami, and others. Secondly they show the possibility of the appearance of pure dicrotic beats at pressures as high as 160—170 mm. Hg., and conclusively demonstrate the dependence of dicrotism in these instances on short, sharp heart systole, for all other factors are constant. Thirdly they demonstrate the possibility of the occurrence of marked dicrotism, even at high pressures, in arteries markedly sclerosed. In the patient, whose tracings are shown in Fig. 6, the vessels were universally tortuous. The postmortem upon the patient, whose tracings are given in Fig. 7, proved the arteries to be markedly sclerosed, with calcareous and atheromatous degeneration, a condition extending into the left radial artery, from which the tracings were taken. These examples disprove the statement of Marey and other writers, that dicrotism does not occur where the vessels are considerably diseased. Similar tracings, showing transition and dicrotic curves on the same line are often met with in mitral disease, where there is irregularity in the force of the heart's contraction. They have also been met with in a patient recovering from pneumonia, Fig. 8; the two tracings were taken within half-an-hour of each other. In this case

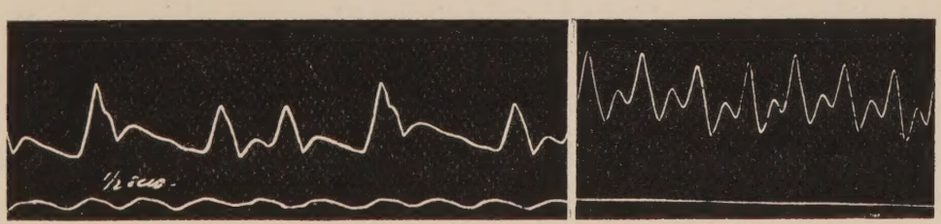


Fig. 8.

there was a remarkable periodicity. The pulse was dicrotic for 5 or 10 minutes, at a pressure of 110—115 mm. Hg.; it would then change to the mixed type, accompanied by a rise of pressure to 125—130 mm. Hg. The interchange from one type to the other was observed over a space of several hours. Fig. 9 shows the effect of amyl nitrite on a normal pulse. It is in agreement with the results of Marey, v. Kries, and

[1] For a further account see Lewis, *Lancet*, 1906.

others. Four portions of the complete tracing are shown. The drug was producing its maximum effect, evidenced by the flushing and throbbing in the vessels, at the third section, where the tracing is lowest on the paper. Dicrotism is abolished at this stage and the pulse is monocrotic; the first beat shown in this section exhibits a predicrotic wave, which is totally absent from the fourth stage, when the pulse is purely dicrotic at a higher pressure. This tracing is of interest in that it shows the impossibility of accepting the view that increased dicrotism depends on lowered pressure; and taken with the observations made upon Figs. 4 and 5, is of interest in showing that the predicrotic likewise is not necessarily abolished by lowered pressure. So far examples

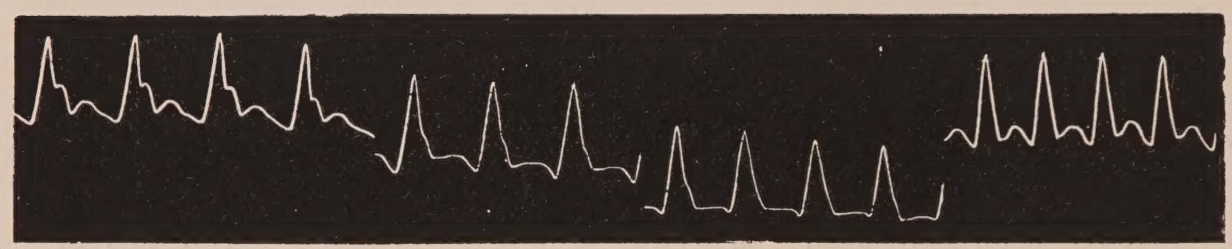


Fig. 9.

of pulses have been given to which the term dicrotic would be applied by all clinicians, pulses dicrotic to the finger and presenting one secondary wave only. It is difficult to fix a standard of dicrotism, there are so many varieties of it, and the definitions of different writers do not agree. Some authorities restrict the term *dicrotic* to pulses which can be recognised as such by palpation. Others hesitate to apply the term to such pulses as exhibit a predicrotic wave. In the numerous and prolonged discussions which have taken place on this subject, much confusion has arisen on this account.

The majority of observers appear to have judged the dicrotic pulses observed from the several clinical standpoints, but have based their hypotheses of the origin, and factors influencing, the dicrotic on a physical definition[1]. There immediately arise several important complications; a pulse may give a typical dicrotic tracing, but the second wave may be so low that the finger does not appreciate it; further a pulse which is palpably dicrotic may show the presence of three waves in a tracing. In Fig. 10 (*a*—*e*), pulse tracings are given from a patient suffering from subacute Bright's disease. The case is of considerable importance, for with a systolic pressure of 180 mm. Hg., the pulse was palpably dicrotic. Tracings from this pulse were taken

[1] Cp. Sanderson, Galabin, Roy and Adami, and others.

at four spring pressures; the pressures given with the tracings were calculated by a method described in previous communications, and are approximately the pressures exerted on the vessel at systole, in mm.Hg. A fifth tracing, *e*, is given showing the effect of obliteration of the radial artery directly below the sphygmograph, to exclude reflected waves from the peripheral radial field. The palpability of the dicrotic and the height of the blood-pressure, were checked by a competent observer. Now this pulse, though palpably dicrotic, demonstrates also a predicrotic wave, and therefore would be excluded from the dicrotic class by certain clinicians. At the same time in considering the factors influencing the prominence of the dicrotic wave in this and other pulses, its value must be expressed from the physical standpoint or fallacy is unavoidable. The only satisfactory definition of dicrotism from this point of view is based on *the relation of the pressures represented in the dicrotic, to those of the primary wave.* It will simplify matters

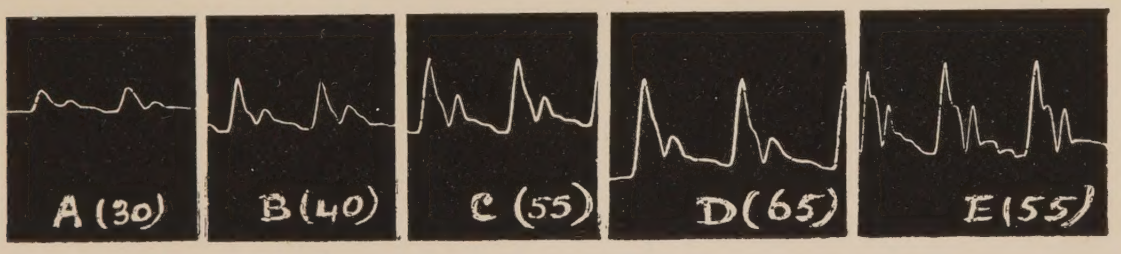


Fig. 10 (*a*—*e*).

if the relation is considered only so far as the highest abscissæ are concerned, though it must be remembered that a pressure wave in travelling may gain or lose in height at the expense or gain of breadth (cp. Galabin). A dicrotic pulse may then be defined as one in which the pressures represented by the dicrotic curve bear a certain minimal relationship to the pressures represented by the primary wave. Unfortunately the dicrotism of the clinician and that of the physicist, as thus defined, do not run hand in hand, neither are they necessarily increased by the same factors. In dealing with this subject there are several considerations of the utmost importance, which have not obtained the attention they deserve. In the first place the dicrotic wave is superimposed on a fundamental wave, which varies in character in different instances. In Fig. 11, the dicrotic wave *A*, has been superimposed by accurate measurement (*ab*, *a′b′*, *a″b″*, are equal throughout the three diagrams), upon three fundamental curves *B*, *C*, and *D* of equal height[1]. From the physical standpoint dicrotism is equal in the

[1] A negative wave preceding the positive wave *A* has been omitted for the sake of greater clearness. Its presence would not affect the questions here considered.

three curves so formed, yet from the clinician's standpoint, the middle curve alone shows increased dicrotism, while in the curve *D* it would be considered decreased. Greater prominence is given to the dicrotic wave according as it approaches the bottom of the outflow curve, and according as its base line tends to the horizontal. In the curve *C*, the dicrotic rise is one-third the height of the primary upstroke; in curve *B*, there is no rise; in curve *D* there is an actual fall. The last curve also suggests a probable source of fallacy in measurements of systolic and diastolic phases of arterial pulse curves; for the dicrotic, or "aortic," notch appears to occur at the point *a'*, whereas in reality it occurs at an

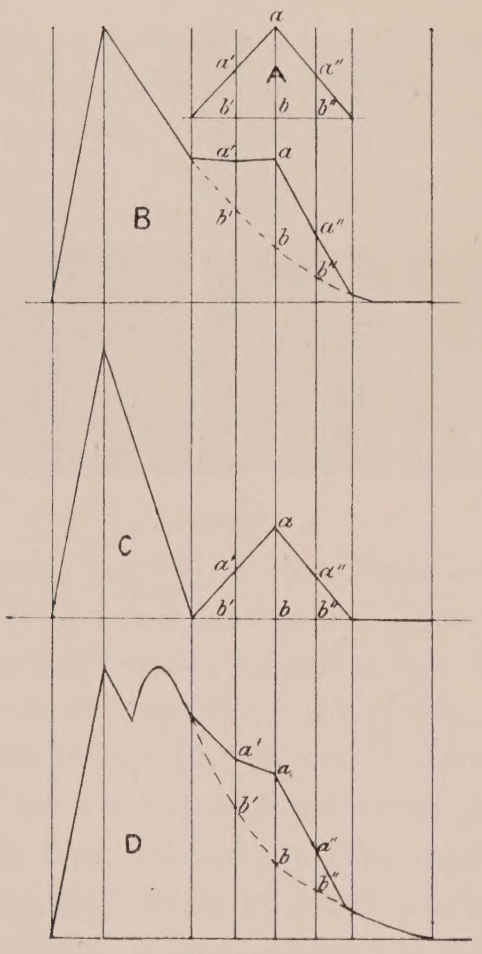


Fig. 11.

earlier time interval. There is a second consideration equally important, but which applies more to the method of recording by air transmission. Marey, Roy, Roy and Adami and others have given figures to show that arteries do not obey Hooke's Law. Recently this has been denied by Haycraft. Nicholls concluded that Hooke's Law is obeyed within a certain limit, but that the limit is exceeded when the tension in the vessel wall overreaches the normal. The difference of opinion appears to depend mainly on the quantitative value of the stretching forces

employed. Accepting the position as laid down by Nicholls, that there is a tendency for the arterial wall to respond by stretching in decreasing amounts as the higher pressures are employed, it is obvious that the expansion of an artery to various pressures may influence the form of the tracing of high tension pulses[1]. For purposes of illustration several curves have been drawn upon a series of lines based on the figure given by Roy and Adami (Fig. 12).

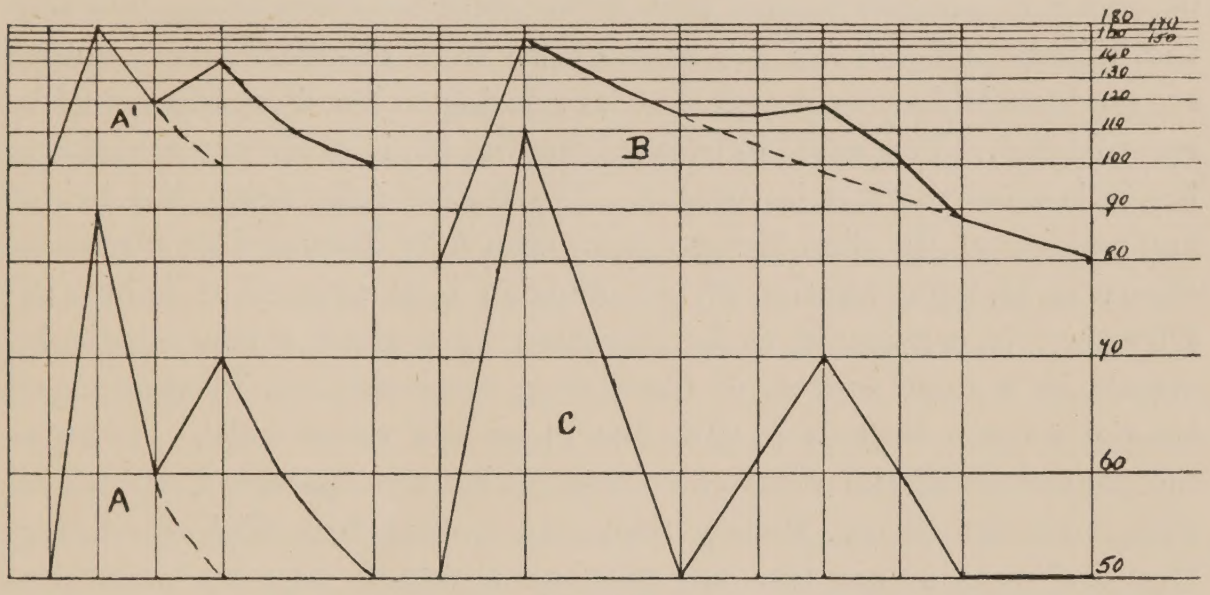


Fig. 12.

The pulse beats *A*, *A'* are of equal proportions, the pressures of the latter are twice those of the former. The upper curve is flattened and dicrotism is decreased in prominence. The "aortic" angle is widened. But as a general rule with higher pressures the fundamental wave is less vertical in its descent. *C* shows the same beat as *C* in Fig. 11; *B* the same beat as *B*, Fig. 11. The two curves have been superimposed on the pressure lines. In *C* the relative amplitude of the dicrotic has been increased by this procedure; in *B* it has been decreased, and the upstrokes of the waves are rendered more oblique. The aortic notch is conspicuously flattened, and the dicrotic wave should therefore be less palpable. Finally if the curves *A* and *C* (Fig. 12) are compared it will be seen that from the physical standpoint dicrotism is more marked in *A* than in *C*, (the primary wave is relatively higher in *C*).

From the arguments which have been employed, the difficulties of gauging the amount of dicrotism will now be apparent, when it is

[1] Dudgeon's Sphygmograph is to some extent defective in that it tends to magnify diastolic phases of the curves; cp. inverted curves. In a previous communication the curtailing of diastolic phases in normal and low pressure tracings was fully entered into (*Practitioner*).

desired to draw conclusions as to the factors influencing the extent of dicrotism, from the evidence lent by sphygmopraph curves. Such conclusions are in the highest degree fallacious, though they have been drawn by some of the most prominent writers on the subject of pulse. So long as the clinician is influenced in determining the state of a patient's circulation by *his*, the clinician's, dicrotism, he is on fairly safe ground, if his inferences are drawn from experience of other cases. The observations made in the first part of this paper are intended to show that the inferences which are generally drawn from the clinician's dicrotism are not invariably correct, and that its relation to blood-pressure must be reconsidered. The case, illustrated in Fig. 10, is perhaps exceptional, but it is none the less important. The factors influencing the height and position of the dicrotic wave have been fully discussed by numerous observers, and the position of the clinician is at present as follows :— Dicrotism is considered to be increased by a short sharp ventricular systole, by a small output of blood from the heart, and by diminished arterial tension, both as regards the blood and vessel wall[1]. So far as the characters of the ventricular contraction are concerned, almost all writers are agreed, (cp. Marey, Sanderson, Galabin, Mahomed etc.). Roy and Adami are the only prominent writers who on theoretical grounds hold an opposite view of the relation of arterial tension to dicrotism. In this respect the observations of this communication and those of a previous contribution fully endorse the view of the last named writers, namely that increased pressure is not necessarily associated with decreased dicrotism. We have seen however that the factors detailed above are the factors which will produce, *par excellence*, the *appearance* of increased dicrotism in a pulse tracing. The main causative element is the rate at which the outflow curve falls, and this depends on the volume and rapidity of output of the ventricular contents and upon the peripheral obstruction. It has been shown that conclusions drawn from actual pulse tracings are open to fallacy; it consequently becomes necessary to make an appeal to the results of experiment on artificial schemata. The only advantage accruing from this study, lies in the simplicity of the apparatus and the possibility of dealing with single and uncomplicated factors. Prominent amongst investigations in this direction, is the work of Marey, Galabin and

[1] Considering the elastic elements of the vessel wall, the blood-pressure, and arterial wall tension will run hand in hand. The two factors may be considered together, for the pulse waves are recorded in the larger vessels where the musculature of the walls plays the least part.

Mahomed. But it would appear that these writers have failed to recognise what at first sight appears somewhat paradoxical, namely that within certain limits the more nearly does the apparatus approach the actual conditions of the circulation, the less positive can be the conclusions drawn from it. In the case of the investigations of the last two observers, no account is given of the method of recording the artificial pulse waves in their flexible tubes. Marey is himself indefinite on the subject, but apparently obtained his records *through* the rubber tubing. The tracings of Galabin show the dicrotic superimposed on a fundamental wave. The fallacies of these methods have been shown. Moreover in the observations of Mahomed[1] and Galabin, the primary wave was imitated by squeezing a rubber bag or dead heart in the hand, and by forcing fluid out of it in this manner into the system of tubing. Under these conditions it is impossible, as it is in the human circulatory system, to make any estimate of the velocity of output at the end of systole, which is granted by almost all observers to be one of the chief elements in determining the amount of the dicrotic. If the pressure in the system is raised by obstructing the outlet, there is a natural tendency for the velocity of output at the end of systole to be decreased, as it is in the case of cardiac muscle; the muscular curve tends to become more isometric. Diminished dicrotism may be due under these circumstances, not to raised pressure, but to decreased velocity, the associated factor. Roy and Adami in their classical researches came to the following conclusions as a result of theoretical considerations and upon the evidence of Valsalva's experiment. "The depth of the dicrotic notch[2] is increased by any cause which diminishes the volume of blood which is thrown out,...and also by any[3] cause which, *cæteris paribus*, raises the pressure within the systemic arteries" (Vol. XLIV. p. 423). Also, "that diminished resistance to the outflow of blood from the arteries, by allowing the outflow to finish at an earlier period in the systole, may lead to marked dicrotism, even though the volume of blood expelled from the heart at each contraction is greater than normal[4]" (Vol. XLIV. p. 425). In view of the conflicting statements and the possible fallacies of such experiments on schemata, a new series of observations has been undertaken, upon a schema of a simple yet, it is believed, satisfactory nature. The schema is represented in Fig. 13.

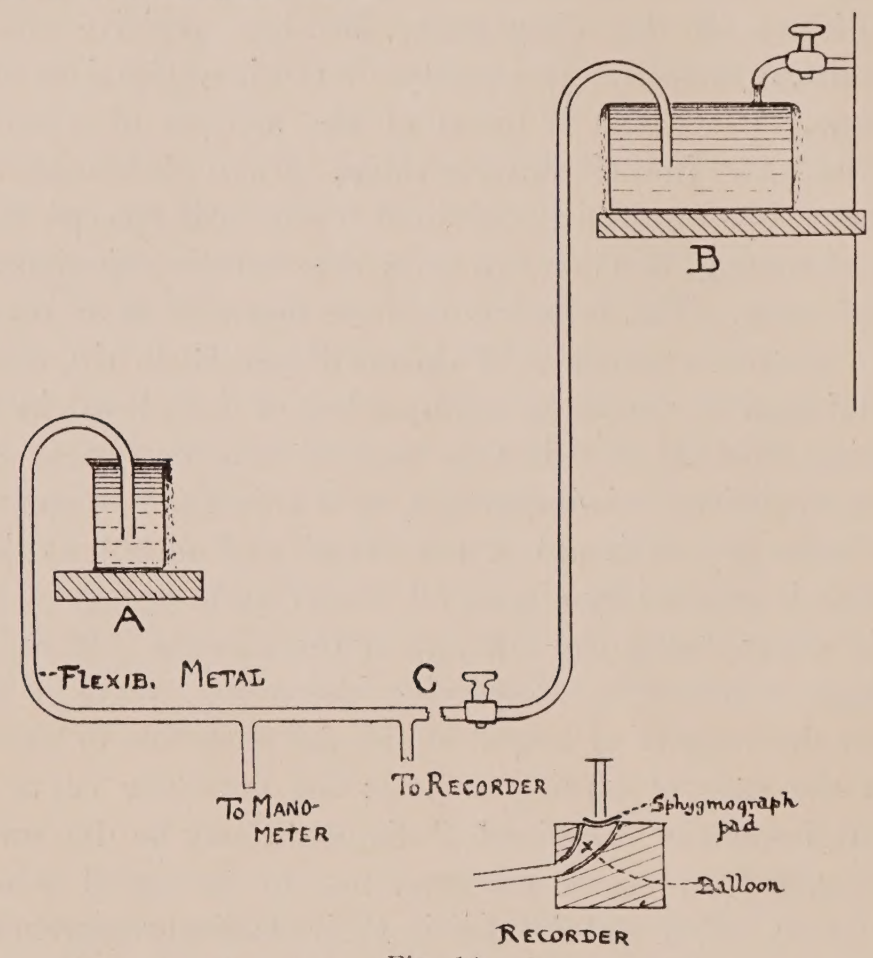
[1] Mahomed was originally of the opinion that increased tension increases dicrotism.

[2] Note that other writers refer to the height of the dicrotic wave.

[3] Reason will be shown for doubting this statement.

[4] Cp. presence of predicrotic on pulses in which the dicrotic is exaggerated (Fig. 10).

The fallacies which have been pointed out are obviated, and the dicrotic wave which is produced stands on a base line and is not superimposed on a fundamental wave.



Fig, 13.

The schema consists, essentially, of two reservoirs filled with water and connected by syphon arrangement composed of rigid tubing[1]. The valves are represented by a glass stopcock, and the horizontal portion of the tube is connected to a manometer and to a recording apparatus. The recording apparatus consists of a small wooden block, in which a flaccid rubber or oiled silk tambour is buried and covered in above by the pad of the sphygmograph, which is fixed to the block. By this means tension in the tambour is avoided, and the pad may be considered to be resting on the surface of the water under pressure. In making an observation the stopcock is opened and adjusted so as to be open to the full. It is then rapidly shut by means of a mechanical

[1] It may be said that rigid tubing is fallacious in that the pressures do not travel as waves, but that the liquid is propelled as a whole. The difference in the production of the dicrotic is mainly a matter of degree, for with flexible tubing there is propulsion of the liquid nearest the valves and along the branches as the primary wave passes. The rigid tubing is essential in that the dicrotic is not superimposed on a fundamental wave.

arrangement, or as rapidly as possible with the hand. In this way constant secondary waves can be obtained. At *C* there is a gap, into which a given length (usually 3 inches to 1 ft.), of glass tubing, oiled silk tubing, rubber tubing, intestine or vessel may be introduced. In an experiment the pressures during the flow, and with the valves shut are estimated, (to the left of the valve in the Fig.). Also the velocity of outflow can be measured by collecting the water falling out of *A*. The pressure with the valve open may be compared to ventricular systolic pressure, assuming the latter to be uniform; the pressure with the valve shut may be compared to arterial diastolic pressure[1]. Systolic pressure can be raised by lifting the block *B*; the velocity of output is thereby increased. The diastolic pressure can be raised by lifting the block *A*; the velocity of output is thereby diminished. The mean pressure can be raised and the velocity kept constant by raising *A* and *B* to a similar extent. The systolic pressure is lowered and velocity of output decreased by opening the stopcock partially. The rate at which the velocity of output is checked can be

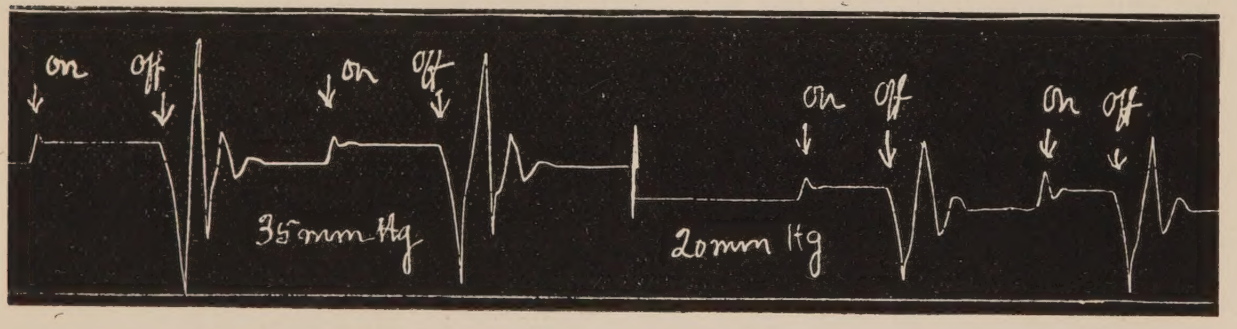


Fig. 14.

varied by altering the rate at which the stopcock is turned. The results obtained with this schema are those which theoretically might be expected. Raising *B*, or lowering *A*, increases the height of the dicrotic as compared with the primary wave. Dicrotism is greatest when the stopcock is at first fully open. The conclusion is that *dicrotism is increased by an increased velocity of output from whatever cause.* If the blocks *A* and *B* are raised to an equal extent, dicrotism is increased in proportion to the rise of mean pressure. An example of this experiment is given in Fig. 14, taken with rigid tubing. The systolic pressures were 35 and 20 mm. Hg. respectively; the diastolic 25 and 10 mm. Hg. respectively. The velocity of outflow in each case

[1] The chief difference between the schema and the circulatory system lies in the fact that the valves are closed in the former by diastolic pressure, and in the latter by the last systolic pressure or first diastolic pressure.

was measured at 14·7 c.c. per sec. The figure shows, first, two waves at the higher pressures, then two waves at the lower pressures. The first two dicrotic waves are approximately double the height of the last two. The primary waves are equal throughout. *A rise of mean pressure may consequently be said to increase dicrotism.* The dicrotic wave is increased if the stopcock is closed more rapidly. *Dicrotism is therefore increased by an increase in the rate at which the outflow through the valves is diminished.* The pressure bottles may be so arranged that with an increased pressure from both dicrotism is increased, in spite of a decrease in the velocity of output; this will be apparent from the instance which is figured[1]. One may conclude that the influence of pressure on dicrotism is a very important one[2]. As the systolic pressures in the largest arteries are proportional in the main to the systolic intraventricular pressures, the conclusions here stated may be applied to the circulatory system. Pressures are the ultimate factors involved in the production of dicrotism; the factor of velocity of output influences dicrotism by controlling the negative pressure (or retardation of the blood particles, as the case may be), developed at the end of systole. Consequently the hypothesis appears reasonable that *in the circulatory system a rise of systolic and a fall of diastolic pressure, other factors remaining constant, leads to increased dicrotism.* We have now to see how this hypothesis accords with other evidence. Galabin and Mahomed found that increased peripheral obstruction in their schemata diminished dicrotism. Reasons have been stated for believing that the velocity of output, and the rate at which it was checked were not constant. Were they constant their results might be explained by assuming a relatively greater rise of diastolic pressure. In Valsalva's experiment the character of the curves indicates rapid output from the heart, and there is reason to believe that peripheral resistance is decreased. In the curves of the second rise, when vasoconstriction is present, output is slower from both heart and peripheral vessels, and dicrotism is decreased[3]. In the case figured (Fig. 10), though systolic pressure was high, velocity of output was rapid, evidenced by the nature of the primary wave, by the moist hot skin of the patient, and by

[1] All experiments so far cited have been performed with rigid and extensible tubing; when the dicrotic was not superimposed on a fundamental wave, the results have been the same in all cases.

[2] Dicrotism is entirely a matter of pressures, and its height is influenced only by pressures. Other factors influencing it effect it through the medium of pressures.

[3] Probably both really and apparently, for the fundamental wave becomes prolonged.

brachial compressions[1]. Thus a high systolic pressure accompanied as it *usually is* by a gradual termination of systole, and by vasoconstriction, is accompanied as a rule by decreased dicrotism both real and apparent[2]. High systolic pressure accompanied as it *may be* by vaso-dilatation, yields increased dicrotism, real and apparent. The occurrence of marked dicrotism in aortic regurgitation[3] may be explained by the disproportion between systolic and diastolic pressures, (cp. Janeway). McVail found it difficult to harmonise the fact that accentuation of the 2nd aortic sound is not invariably accompanied by increased dicrotism. The explanation is I believe to be found in the views set forth in the description of Figs. 11 and 12.

Marey, Mahomed and Galabin found that dicrotism is increased by the use of less rigid tubing, and concluded that increased expansibility and decreased tension of the arterial wall favour dicrotism[4]. That they produce an apparent increase is quite possible, but as Roy and Adami considered, the reverse is actually the case; the more rigid the walls and valves the greater the dicrotism. Tension in the arterial wall will not be considered; it runs hand in hand with pressure; we are merely concerned with the extensibility and elasticity of the tube. Eventually the subject reduces itself to a question of expansibility, given that the tube is elastic. A rigid tube gives a dicrotic wave

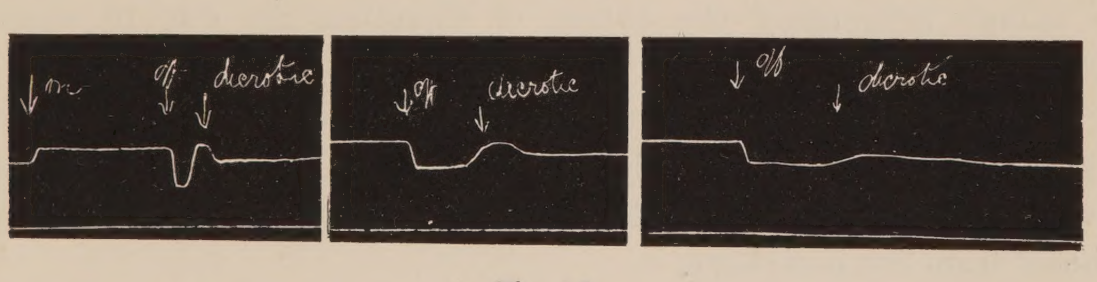


Fig. 15.

higher than the primary wave, a fact well known to many engineers who have had experience of simple force pumps. The great local rise of pressure in the pipes of a water supply, upon rapidly shutting a tap is well recognised (see also Fig. 14). In experiments on the schema, the introduction of flexible tubing, whether elastic or not, produces a marked decrease of dicrotism. In Fig. 15 the result of introducing $\frac{1}{2}$, $1\frac{1}{2}$ and 3 inches of chicken gut into the gap *C* is shown. The tube as a

[1] Cp. Hill, Barnard and Sequeira, and Lewis, *Practitioner*.

[2] To what extent it is real and to what extent apparent it is impossible to say.

[3] The reflection being from the widening arterial walls, and therefore chiefly at the origin of the branches (cp. Lewis, *Lancet*).

[4] The same view is held by several leading physiologists to-day.

whole was maintained of constant length ; the pressures employed and the velocity of outflow were also kept constant. The dicrotic is greatly decreased in height, and at the same time delayed and spread out over a larger interval (cp. Keyt). The same results were obtained with thin rubber, artery, and oiled silk. Fig. 9 shows dicrotism abolished where the arterial walls are most flaccid[1].

The results obtained with the schema employed are possibly open to criticism. Bearing this in mind, the arrangement of the tubing and its several branches and outlets have been varied. Also flexible metal tubing has been exchanged for glass, and glass for rubber of various sizes and thicknesses, also the diameter of the outlet as compared to the inlet has been varied. In the main the results have been in accordance with those set forth in the above pages.

Summary of Chief Conclusions.

1. A patient with a systolic blood-pressure of 180 mm. Hg., may have a pulse palpably dicrotic. The usual range of pressures where the pulse is purely dicrotic is from 80—125 mm. Hg.

2. There is an important distinction between apparent (clinical) and real (physical) increased dicrotism. Many of the statements as to the factors influencing the extent of dicrotism are due to want of recognition of this fact.

3. The chief factors involved in the production of *apparent* increased dicrotism are low blood-pressure and its usual accompaniments, short sharp heart systole, flaccid arterial walls, and rapid output of blood from the periphery.

4. The chief factors involved in the production of *real* increased dicrotism are those factors which usually accompany low blood-pressure, with the exception of flaccid arterial walls; ultimately all factors involved may be reduced to pressures.

5. A rise of systolic or a fall of diastolic pressure tends, with other factors constant, to increase dicrotism. An equal rise of both systolic and diastolic pressure, with other factors constant, tends to increase dicrotism.

6. Rigidity of the arterial walls tends to increase dicrotism, other factors being equal.

[1] Conclusions from such observations, have to be very carefully drawn, as the factors involved are never single but always complex.

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[1] The last two papers referred to are in the hands of the printers.